IN THE SPECIFICATION

ABSTRACT

Please amend the **Abstract** as follows:

The present invention provides a method of reducing the period within which a plant's natural defence mechanism responds to attack by a plant pathogen, the method comprising causing the plant to maintain, in at least a part of the plant, a level of BiP which is greater than the endogenous level for said plant in non-stressful conditions. Increased BiP levels can be achieved by transformation with a nucleic acid encoding BiP or calreticulin or modifying signal transduction pathways leading to BiP induction. BiP levels can be increased above endogenous levels by over-expressing BiP or calreticulin. The invention also provides for a modified plant produced by the method of the invention.

DETAILED DESCRIPTION OF THE INVENTION

Example 6

Please amend the paragraph at page 17, line 33 through page 18, line 7 with the following amended paragraph.

To compare the CDE- and UPR-mediated induction of BiP, we wanted to test if both stimuli are additive. For this purpose, we prepared protoplasts which are known to exhibit induced levels of ß-1,3-glucanase (Denecke et al., 1995). This is not surprising as protoplasts are prepared with CDEs. These protoplasts were then treated with tunicamycin, to superimpose the UPR onto the CDE response. Figure [[7]] 6 shows that both stimuli are additive, exhibited by a further induction of BiP by tunicamycin. This suggests that both mechanisms are different. Interestingly, ß-1,3-glucanase expression is inhibited by tunicamycin. The additional ER stress could trap BiP in malfolded protein complexes, thus making it unavailable to promote PR protein synthesis on the rough ER. The results suggested that although BiP induction alone is not sufficient to trigger PR protein synthesis, sufficient BiP levels are required to promote PR gene expression.